(FILE 'HOME' ENTERED AT 08:08:40 ON 25 SEP 2001)

FILE 'MEDLINE' ENTERED AT 08:08:52 ON 25 SEP 2001

L1	427	S	POOLED SAMPLE#
L2	2	S	L1 AND STOOL
L3	8861	S	PATIENT POPULATION
L4	427	S	POOLED SAMPLE#
L5	0	S	L3 AND L4
L6	21985	S	P53
L7	9124	S	ENERGY TRANSFER
L8	22	S	L6 AND L7
L9	230226	S	MUTATION#
L10	12	S	L8 AND L9
L11	1314	S	P53 AND (POINT (W) MUTATION)
L12	2	S	ENERGY RESONANCE TRANSFER
L13	0	S	L11 AND RHODAMINE
L14	0	S	L11 AND FLUORESCEIN
L15	611	S	L11 AND DETECT?
L16	0	S	L11 AND PRIMER EXTENSION
L17	36	S	L15 AND PROBE#
L18	92	S	L15 AND PRIMER#
L19	8	S	L17 AND RAS
L20	22	S	P53 AND (ENERGY TRANSFER)
L21	11	S	P53 AND RHODAMINE
L22	1	S	L21 AND FLUORESCEIN
L23	0	S	P53 AND FRET
L24	4	S	RAS AND FRET
L25	1	S	POOL(4A)PATIENT(4A)SAMPLE?
L26	6	S	POOL?(4A)STOOL(4A)SAMPLE?

FILE 'CAPLUS' ENTERED AT 08:28:47 ON 25 SEP 2001

L27	3	S	L26
L28	4	S	L23
L29	0	S	L5
L30	57	S	POOL? AND STOOL?
L31	0	S	MUTATION AND L30
L32	1	S	DNA AND L30
L33	681	S	FRET
L34	1261	S	FLUORESCEN?(2A)ENERGY(2A)RESONANCE(2A)TRANSFER
L35	1417	S	L33 OR L34
L36	1	S	L35 AND (STOOL OR EXCRET?)
L37	7	S	L35 AND POOL?
L38	4634	S	POPULATION AND SCREEN?
L39	2	S	L35 AND L38
L40	516	S	POPULATION(2A)SCREEN?
L41	187	S	MUTATION? AND L40
L42	1127941	S	SAMPLE?
L43	43	S	L41 AND L42
L44	41	S	L40 AND (POOL? OR COMBINE OR COMBINING OR COMBINED)
L45	10	S	L44 AND SAMPLE?
L46	1436	S	POOL?(2A)SAMPLE?
L47	0	S	L46 AND FRET
L48	4	S	L46 AND STOOL
L49	0	S	L48 AND DNA
L50	0	S	L48 AND MUTATION
L51	10	S	L45
L52	4	S	L48

L28 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2001 ACS
AN 1998:307053 CAPLUS
DN 129:37221
TI Detection of point mutation using double fluorescent-labeled probes and detection of gene abnormalities by the method
IN Hirano, Kenichi
PA Hamamatsu Photonics K. K., Japan
SO Jpn. Kokai Tokkyo Koho, 14 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 10127300	A2	19980519	JP 1996-290235	19961031

AB Point mutation in a specific sequence of target nucleic acids is detected by hybridizing the target nucleic acids with a complementary probe labeled with a fluorescent dye at one end and another fluorescent dye with the other end, both dyes show fluorescence resonance energy transfer (**FRET**) between them, at a higher temp. than room temp. and measuring the ratio of fluorescence intensity of one fluorescent dye to that of the other dye at the max. absorption wavelengths. Gene abnormalities, e.g. of oncogenes, are detected by the above method. **FRET** efficacy is dependent on temp. and fluorescence intensity is measured in various temp. points. For example, mutations in the gene **p53** of liver samples were detected.

The diagnosis of old and new gastrointestinal parasites.

AU Long E G; Christie J D
CS Division of Bacterial and Mycotic Diseases, Centers for Disease Control and Prevention, Atlanta, Georgia, USA.

SO CLINICS IN LABORATORY MEDICINE, (1995 Jun) 15 (2) 307-31. Ref: 140
Journal code: DLS; 8100174. ISSN: 0272-2712.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199510
ED Entered STN: 19951026
Last Updated on STN: 19951026
Entered Medline: 19951019

AB For the foreseeable future, light microscopy will continue to be the method of choice for diagnosing gastrointestinal parasites. However, in selected circumstances, the use of commercially available immunofluorescent kits will supersede the use of routine light microscopy for diagnosis of Giardia lamblia and Cryptosporidium parvum. These techniques may used to diagnose invasive amebic infections caused by E. histolytica in the future. **Pooling stool samples** from the same or even different patients may offer a means to process specimens in a more efficient and cost-effective manner without lowering the predictive value of an ova and parasite examination. Although we suggest that, with some exceptions, stools for ova and parasite examination should not be accepted past the fourth day of hospitalization, we cannot recommend the use of a single stool sample for diagnosis without extensive studies in individual parasitology laboratories. Techniques have still not been developed for the optimum methods of concentration of stool for diagnosis of coccidian infections. For most laboratories, the diagnosis of microsporidian infections remains problematic because of the lack of a commercial source for oocysts to provide positive control material. (Note: There is now a commercial source for oocysts available.)

45 ANSWER 6 OF 10 CAPLUS COPYRIGHT 2001 ACS
AN 1999:402039 CAPLUS
DN 131:40257
TI Applications of constant denaturant capillary electrophoresis/high-fidelity polymerase chain reaction to human genetic analysis
AU Li-Sucholeiki, Xiao-Cheng; Khrapko, Konstantin; Andre, Paulo C.; Marcelino, Luisa A.; Karger, Barry L.; Thilly, William G.
CS Division Bioengineering Environmental Health, Center Environmental Health Sciences, Massachusetts Institute Technology, Cambridge, MA, 02142, USA
SO Electrophoresis (1999), 20(6), 1224-1232
CODEN: ELCTDN; ISSN: 0173-0835
PB Wiley-VCH Verlag GmbH
DT Journal; General Review
LA English
AB A review is given on the author's own works with 32 refs. Const. denaturant capillary electrophoresis (CDCE) permits high-resoln. sepn. of single-base variations occurring in an 100 bp isomelting DNA sequence based on their differential melting temps. By coupling CDCE for highly efficient enrichment of mutants with high-fidelity PCR (hifi PCR), the authors developed an anal. approach to detecting point mutations at frequencies .gtoreq.10(tm) in human genomic DNA. The authors present several applications of this approach in human genetic studies. The authors have measured the point mutational spectra of a 100 bp mitochondrial DNA sequence in human tissues and cultured cells. The observations have led to the conclusion that the primary causes of mutation in human mitochondrial DNA are spontaneous in origin. In the course of studying the mitochondrial somatic mutations, the authors have also identified several nuclear pseudogenes homologous to the analyzed mitochondrial DNA fragment. Recently, through developments of the means to isolate the desired target sequences from bulk genomic DNA and to increase the loading capacity of CDCE, the authors have extended the CDCE/hifi PCR approach to study a chem. induced mutational spectrum in a single-copy nuclear sequence. Future applications of the CDCE/hifi PCR approach to human genetic anal. include studies of somatic mitochondrial mutations with respect to aging, measurement of mutational spectra of nuclear genes in healthy human tissues and **population screening** for disease-assocd. single nucleotide polymorphisms (SNPs) in large **pooled samples**.
RE.CNT 32

L17 ANSWER 35 OF 36 MEDLINE
AN 91183169 MEDLINE
DN 91183169 PubMed ID: 2009369
TI Mutation of the **p53** gene in human acute myelogenous leukemia.
AU Slingerland J M; Minden M D; Benchimol S
CS Ontario Cancer Institute, Toronto, Canada.
SO BLOOD, (1991 Apr 1) 77 (7) 1500-7.
Journal code: A8G; 7603509. ISSN: 0006-4971.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199105
ED Entered STN: 19910526
Last Updated on STN: 19970203
Entered Medline: 19910506
AB Heterogeneity of **p53** protein expression is seen in blast cells of patients with acute myelogenous leukemia (AML). **p53** protein is **detected** in the blasts of certain AML patients but not in others. We have identified **p53** protein variants with abnormal mobility on gel electrophoresis and/or prolonged half-life (t 1/2). We have sequenced the **p53** coding sequence from primary blast cells of five AML patients and from the AML cell line (OCIM2). In OCIM2, a **point mutation** in codon 274 was identified that changes a valine residue to aspartic acid. A wild type **p53** allele was not **detected** in these cells. Two point mutations (codon 135, cysteine to serine; codon 246, methionine to valine) were identified in cDNA from blasts of one AML patient. Both mutations were present in blast colonies grown from single blast progenitor cells, indicating that individual leukemia cells had sustained mutation of both **p53** alleles. The cDNAs sequenced from blast samples of four other patients, including one with prolonged **p53** protein t 1/2 and one with no **detectable p53** protein, were fully wild type. Thus, the heterogeneity of **p53** expression cannot be explained in all cases by genetic change in the **p53** coding sequence. The prolonged t 1/2 of **p53** protein seen in some AML blasts may therefore reflect changes not inherent to **p53**. A model is proposed in which mutational inactivation of **p53**, although not required for the evolution of neoplasia, would confer a selective advantage, favoring clonal outgrowth during disease progression.

N 94031784 PubMed ID: 8217795
TI Occurrence of point mutations in **p53** gene is not increased in patients with acute myeloid leukaemia carrying an activating N-**ras** mutation.
AU Buhler-Leclerc M; Gratwohl A; Senn H P
CS Institut fur Medizinische Mikrobiologie, Universitat Basel, Switzerland.
SO BRITISH JOURNAL OF HAEMATOLOGY, (1993 Jul) 84 (3) 443-50.
Journal code: AXC; 0372544. ISSN: 0007-1048.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199312
ED Entered STN: 19940117
Last Updated on STN: 19940117
Entered Medline: 19931207
AB The frequency of simultaneously **detecting** N-**ras** and **p53** gene mutations was studied in leukaemia cells of patients with acute myeloid leukaemia (AML) or with myelodysplastic syndrome (MDS). Using in vitro DNA amplification followed by oligonucleotide hybridization analysis, 45 AML and six MDS patients were screened for activating mutations in codons 12, 13 and 61 of N-**ras**. Ten of them (eight AML and two MDS) were found positive. These 10 patients and 10 others without activating N-**ras** mutation were further analysed by direct sequencing of the amplified exons for **p53** mutations and for atypical N-**ras** mutations. Beside the activating mutations in the N-**ras** gene, no additional transforming or nontransforming mutations could be **detected** in the N-**ras**. However, exon 7 of **p53** was mutated in two AML patients without activating N-**ras** mutation. These data show that **p53** mutations occurred with half the frequency of N-**ras** mutations in AML and that no positive correlation could be found between the onset of mutations in N-**ras** and **p53** genes.